Potential bioterrorism agent: Category A

BOTULISM *(foodborne, wound and infant)*

DISEASE REPORTING

In Washington

DOH receives 0 to 2 reports of foodborne botulism per year, 0 to 2 reports of wound botulism per year, and 0 to 4 reports of infant botulism per year. The last death in Washington associated with botulism occurred in 1983.

Recent foodborne botulism cases in Washington were associated with improperly home-canned asparagus, beets, corn, carrots, spinach, and salsa. Wound botulism is most frequently associated with injection drug use, particularly black tar heroin.

Purpose of reporting and surveillance

- To assist in the diagnosis and treatment of cases.
- To classify reported cases as foodborne, infant, or wound botulism.
- For foodborne botulism, to identify sources of transmission (e.g., contaminated foods) and to prevent further transmission from such sources.
- For foodborne botulism, to identify others who have eaten the contaminated food(s) and to provide counseling.
- To raise the index of suspicion of a possible bioterrorism event if no natural exposure source is identified.

Reporting requirements

- Health care providers: **immediately notifiable to Local Health Jurisdiction**
- Hospitals: **immediately notifiable to Local Health Jurisdiction**
- Laboratories: No requirements for reporting
- Local health jurisdictions: **suspected or confirmed cases are immediately notifiable to DOH Communicable Disease Epidemiology: 1-877-539-4344**

CASE DEFINITION FOR SURVEILLANCE

FOODBORNE BOTULISM

Clinical criteria for diagnosis

Ingestion of botulinum toxin results in an illness of variable severity. Common symptoms are diploplia, blurred vision, and bulbar weakness. Symmetric paralysis may progress rapidly.

Laboratory criteria for diagnosis

- Detection of botulinum toxin in serum, stool, or patient's food or
- Isolation of *Clostridium botulinum* from stool.

Case definition

- Probable: A clinically compatible case with an epidemiologic link (e.g., ingestion of a home-canned food within the previous 48 hours).
- Confirmed: A clinically compatible case that is laboratory confirmed or that occurs among persons who ate the same food as persons who have laboratory-confirmed botulism.

INFANT BOTULISM

Clinical criteria for diagnosis

An illness of infants, characterized by constipation, poor feeding, and "failure to thrive" that may be followed by progressive weakness, impaired respiration, and death.

Laboratory criteria for diagnosis

- Detection of botulinum toxin in serum or stool or
- Isolation of *Clostridium botulinum* from stool.

Case definition

- Confirmed: a clinically compatible case that is laboratory-confirmed, occurring in a child aged < 1 year.

WOUND BOTULISM

Clinical criteria for diagnosis

An illness resulting from toxin produced by *Clostridium botulinum* that has infected a wound. Common symptoms are diplopia, blurred vision, and bulbar weakness. Symmetric paralysis may progress rapidly.

Laboratory criteria for diagnosis

- Detection of botulinum toxin in serum or
- Isolation of *Clostridium botulinum* from wound.

Case definition

- Confirmed: a clinically compatible case that is laboratory confirmed in a patient who has no suspected exposure to contaminated food and who has a history of a fresh, contaminated wound during the 2 weeks before onset of symptoms.

A. DESCRIPTION

1. Identification

There are three forms of botulism-foodborne (the classic form), wound and intestinal (infant and adult) botulism. The site of toxin production is different for each of the forms but all share the flaccid paralysis that results from botulinum neurotoxin. Intestinal botulism has been proposed as the new designation for what had been called infant botulism. This new name has not been officially accepted as of mid-1999, but will be generally used in this chapter instead of infant botulism.

Foodborne botulism is a severe intoxication resulting from ingestion of preformed toxin present in contaminated food. The illness is characterized by acute bilateral cranial nerve impairment and descending weakness or paralysis. Visual difficulty (blurred or double vision), dysphagia and dry mouth are often the first complaints. These symptoms may extend to a symmetrical flaccid paralysis in a paradoxically alert person. Vomiting and constipation or diarrhea may be present initially. Fever is absent unless a complicating infection occurs. The case-fatality rate in the US is 5%-10%. Recovery may take months.

In wound botulism the same clinical picture is seen after the causative organism contaminates a wound in which anaerobic conditions develop.

Intestinal (infant) botulism is the most common form of botulism in the US; it results from ingestion of *Clostridium botulinum* spores with subsequent outgrowth and in-vivo toxin production in the large intestine. It affects infants under 1 year of age almost exclusively, but can affect adults who have altered GI anatomy and microflora. The illness typically begins with constipation, followed by lethargy, listlessness, poor feeding, ptosis, difficulty swallowing, loss of head control, hypotonia extending to generalized weakness (the “floppy baby”) and, in some cases, respiratory insufficiency and arrest. Infant botulism has a wide spectrum of clinical severity, ranging from mild illness with gradual onset to sudden infant death; some studies suggest that it may cause an estimated 5% of cases of sudden infant death syndrome (SIDS). The case-fatality rate of hospitalized cases in the US is less than 1%; without access to hospitals with pediatric intensive care units, more would die.

Diagnosis of foodborne botulism is made by demonstration of botulinum toxin in serum, stool, gastric aspirate or incriminated food; or by culture of *C. botulinum* from gastric aspirate or stool in a clinical case. Identification of organisms in a suspected food is helpful but not diagnostic because botulinum spores are ubiquitous; the presence of toxin in a suspected contaminated food source is more significant. The diagnosis may be accepted in

a person with the clinical syndrome who had consumed a food item incriminated in a laboratory confirmed case. Wound botulism is diagnosed by toxin in serum or by positive wound culture. Electromyography with rapid repetitive stimulation can be useful in corroborating the clinical diagnosis for all forms of botulism.

The diagnosis of intestinal botulism is established by identification of *C. botulinum* organisms and/or toxin in patient's feces or in autopsy specimens. Toxin is rarely detected in the sera of patients.

2. Infectious Agent

Foodborne botulism is caused by toxins produced by *Clostridium botulinum*, a spore forming obligate anaerobic bacillus. A few nanograms of the toxin can cause illness. Most human outbreaks are due to types A, B, E and rarely to type F. Type G has been isolated from soil and autopsy specimens but an etiologic role in botulism has not been established. Type E outbreaks are usually related to fish, seafood and meat from marine mammals.

Toxin is produced in improperly processed, canned, low acid or alkaline foods, and in pasteurized and lightly cured foods held without refrigeration, especially in airtight packaging. The toxin is destroyed by boiling; inactivation of spores requires much higher temperatures. Type E toxin can be produced slowly at temperatures as low as 3ºC (37.4ºF), which is lower than that of ordinary refrigeration.

Most cases of infant botulism have been caused by type A or B. A few cases (toxin types E and F) have been reported from neurotoxigenic clostridial species *C. butyricum* and *C. baratii*, respectively.

3. Worldwide Occurrence

Sporadic cases, family and general outbreaks occur where food products are prepared or preserved by methods that do not destroy the spores and permit toxin formation. Cases rarely result from commercially processed products; outbreaks have occurred from contamination through cans damaged after processing. Cases of intestinal botulism have been reported from five continents: Asia, Australia, Europe, and North and South America. The actual incidence and distribution of intestinal botulism are unknown because physician awareness and diagnostic testing remain limited, as demonstrated by a review of intestinal botulism cases reported between 1976, when it was first recognized in California, and the beginning of 1999. Of the 1,700 cumulative global case total, over 1,400 were reported by the US, with close to half of those cases reported by California. Internationally, about 150 cases have been detected in Argentina; less than 20 each in Australia and Japan; less than 15 in Canada; and about 30 from Europe (mostly Italy and the UK), with scattered reports from Chile, China, Israel and Yemen.

4. Reservoir

Spores are ubiquitous in soil worldwide; they are frequently recovered from agricultural products, including honey. Spores are also found in marine sediments and in the intestinal tract of animals, including fish.

5. Mode of Transmission

Foodborne botulism is acquired by ingestion of food in which toxin has been formed, predominantly after inadequate heating during preservation and without subsequent adequate cooking. Most poisonings in the US are due to home canned vegetables and fruits; meat is an infrequent vehicle. Several outbreaks have recently occurred following consumption of uneviscerated fish. Cases associated with baked potatoes and improperly handled commercial potpies have been reported. One recent outbreak was attributed to sautéed onions, two others to minced garlic in oil. Some of these recent outbreaks originated in restaurants. Newer varieties of certain garden foods such as tomatoes, formerly considered too acidic to support growth of *C. botulinum*, may no longer be low hazard foods for home canning.

In Canada and Alaska, outbreaks have been associated with seal meat, smoked salmon and fermented salmon eggs. In Europe, most cases are due to sausages and smoked or preserved meats; in Japan, to seafood. These differences have been attributed in part to the greater use of sodium nitrite for preserving meats in the US.

Wound botulism cases often result from contamination of the wounds by ground-in soil or gravel or from improperly treated open fractures. Wound botulism has been reported among chronic drug abusers (primarily in dermal abscesses from subcutaneous injection of heroin and also from sinusitis in cocaine sniffers).

Intestinal botulism arises from ingestion of botulinum spores that then germinate in the colon, rather than by ingestion of preformed toxin. Possible sources of spores for infants are multiple, and include foods and dust. Honey, fed on occasion to infants, can contain *C. botulinum* spores.

6. Incubation period

Neurologic symptoms of foodborne botulism usually appear within 12-36 hours, sometimes several days, after eating contaminated food. In general, the shorter the incubation period, the more severe the disease and the higher the case-fatality rate. The incubation period of intestinal botulism in infants is unknown, since the precise time that the infant ingested the causal botulinum spores cannot be determined.

7. Period of communicability

Despite excretion of *C. botulinum* toxin and organisms at high levels (ca. 10^6 organisms/g) in the feces of intestinal botulism patients for weeks to months after onset of

illness, no instance of secondary person to person transmission has been documented. Foodborne botulism patients typically excrete the toxin and organisms for shorter periods.

8. Susceptibility and resistance

Susceptibility is general. Almost all patients hospitalized with intestinal botulism have been between 2 weeks and 1 year of age; 94% were less than 6 months, and the median age at onset was 13 weeks. Cases of intestinal botulism have occurred in all major racial and ethnic groups. Adults with special bowel problems leading to unusual GI flora (or with a flora unintentionally altered by antibiotic treatment for other purposes) may be susceptible to intestinal botulism.

B. METHODS OF CONTROL

1. Preventive measures:

a. Ensure effective control of processing and preparation of commercially canned and preserved foods.
b. Educate those concerned with home canning and other food preservation techniques regarding the proper time, pressure and temperature required to destroy spores, the need for adequately refrigerated storage of incompletely processed foods, and the effectiveness of boiling, with stirring, home canned vegetables for at least 10 minutes to destroy botulinum toxins.
c. *C. botulinum* may or may not cause container lids to bulge and the contents to have "off-odors." Other contaminants can also cause cans or bottle lids to bulge. Bulging containers should not be opened, and foods with off-odors should not be eaten or taste tested. Commercial cans with bulging lids should be returned unopened to the vendor.
d. Although *C. botulinum* spores are ubiquitous, identified sources such as honey, should not be fed to infants.

2. Control of patient, contacts and the immediate environment:

a. Report to local health authority.
b. Isolation: Not required, but handwashing is indicated after handling soiled diapers.
c. Concurrent disinfection: The implicated food(s) should be detoxified by boiling before discarding, or the containers broken and buried deeply in soil to prevent ingestion by animals. Contaminated utensils should be sterilized by boiling or by chlorine disinfection to inactivate any remaining toxin. Usual sanitary disposal of feces from infant cases. Terminal cleaning.
d. Quarantine: None.
e. Management of contacts: None for simple direct contacts. Those who are known to have eaten the incriminated food should be purged with cathartics, given gastric lavage and high enemas and kept under close medical observation. The decision to provide presumptive treatment with polyvalent (equine AB or ABE) antitoxin to

asymptomatic exposed individuals should be weighed carefully: balance the potential protection when antitoxin is administered early (within 1-2 days after eating the implicated meal) against the risk of adverse reactions and sensitization to horse serum.

f. Investigation of contacts and source of toxin: Study recent food history of those ill, and recover all suspected foods for appropriate testing and disposal. Search for other cases of botulism to rule out foodborne botulism.
g. Specific treatment for foodborne and wound botulism: Intravenous administration as soon as possible of 1 vial of polyvalent (AB or ABE) botulinum antitoxin, available from CDC, Atlanta, through DOH Communicable Disease Epidemiology (1-877-539-4344 or 1-877-539-4344) is considered a part of routine treatment (the emergency telephone number at CDC for botulism calls during regular office hours is 404-639-2206; and after hours and on weekends is 404-639-2888). Serum should be collected to identify the specific toxin before antitoxin is administered, but antitoxin should not be withheld pending test results. Most important is immediate access to an intensive care unit so that respiratory failure, the usual cause of death, can be anticipated and managed promptly. For wound botulism, in addition to antitoxin, the wound should be debrided and/or drainage established, and appropriate antibiotics (e.g., penicillin) administered. For infant botulism: intramuscular injection of human-derived botulinum antitoxin (formerly known as botulinum immune globulin) should be used, and can be obtained through the California Department of Health Services 24 hours/day (510-540-2646). See also: Arnon SS, Schechter R, Inglesby TV, et al. Botulinum toxin as a biological weapon: medical and public health management. JAMA 2001; 285:1059–1070 (in *Additional Resources*).

3. Epidemic measures

Suspicion of a single case of botulism should immediately raise the question of a group outbreak involving a family or others who have shared a common food. Home preserved foods should be the prime suspect until ruled out, although restaurant foods or widely distributed commercially preserved foods are occasionally identified as the source of intoxication and pose a far greater threat to the public health.

In addition, because recent outbreaks have implicated unusual food items, even theoretically unlikely foods should be considered. When any food is implicated by epidemiologic or laboratory findings, immediate recall of the product is necessary, as is immediate search for people who shared the suspected food and for any remaining food from the same source. Any remaining food may be similarly contaminated; such food, if found, should be submitted for laboratory examination. Sera, gastric aspirates and stool from patients and (when indicated) from others exposed but not ill should be collected and forwarded immediately to a reference laboratory before administration of antitoxin.

4. International measures

Commercial products may have been distributed widely; international efforts may be required to recover and test implicated foods. International common source outbreaks have occurred.

5. Bioterrorism measures

Botulinum toxins can be easily used by terrorists. Although the greatest threat may be via aerosol use, the more common threat may be via its use in food and drink. The occurrence of even a single case of botulism, especially if there is no obvious source of an improperly preserved food should raise the possibility of deliberate use of botulinum toxin. All such cases should be reported immediately so that appropriate investigations can be initiated without delay.